

population, while SoxB2 proteins have been proposed to control the progression from stem cell to neuron. Although there has been intense study of the function of SoxB1 proteins, less is known about the SoxB2 group. Prior work in chick embryos suggests that Sox21, a SoxB2 protein, regulates the progression from progenitor cell to neuron by counteracting the activity of SoxB1 proteins. To better understand the role of Sox21 during neurogenesis, our lab has cloned the *Xenopus laevis* ortholog of Sox21 (*Xlsox21*). We found that overexpression of *Xlsox21* results in a phenotype that differs greatly from that in chick. Rather than suppressing the activity of SoxB1 proteins, we found that *Xlsox21* enhances the expression of SoxB1 proteins thereby maintaining progenitors in an undifferentiated state. We also found that it represses neurogenesis by preventing progenitors from further progression and differentiation. Together these results suggest that *Xlsox21* plays a different role than previously thought. With this we plan to further examine the role of *Xlsox21* by gain and loss of function analyses and observing effects on the development of structures known to express *Xlsox21*, including the olfactory placode and the midbrain–hindbrain boundary.

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Program/Abstract # 139

Arx regulates the cell cycle in cortical ventricular zone progenitor cells

Jacqueline Simonet^a, Ginam Cho^b, Jeffrey A. Golden^{a,b}

^aDept. of Cell and Dev. Biol., Univ. of Penn, Phila., PA, USA

^bDept. of Path., Univ. of Penn, Phila., PA, USA

Aristaless-related homeobox gene (*Arx*) is mutated in many epilepsy and mental retardation syndromes, including West syndrome, Partington syndrome, and a spectrum of X-linked mental retardation disorders. Many different mutations of *Arx* have been documented, missense mutations and polyalanine expansions, and even patients with the same mutation show phenotypic heterogeneity suggesting that *Arx* may have many different functions in development. During development *Arx* is expressed in progenitor cells throughout the forebrain. In the pallium *Arx* is expressed in the proliferative ventricular zone (VZ) where the excitatory neurons of the cortex are born. *Arx*−/Y mice show a decreased proliferation in the VZ of the cortex resulting in smaller brains (Kitamura et al, 2002). Friocourt et al. (2008) have shown that overexpression of *Arx* in the VZ of the cortex increases cell cycle length. However, the effect on the cell cycle of the loss of *Arx* in the VZ has not been examined. In addition, how *Arx* regulates the cell cycle and therefore the pattern of differentiation of the neuronal progenitor cells in the VZ is not well understood. We examined the effect of the specific loss of *Arx* in the VZ of the cortex on the cell cycle of the neuronal progenitor cells through IdU/BrdU pulse experiments and examination of layer formation of the cortex. We have determined that *Arx* has Pax6 independent influences on the cell cycle. Furthermore, we find a lengthening of the cell cycle that results in a decrease in differentiated excitatory neurons in the cerebral cortex.

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Program/Abstract # 140

Visualization of CiPax6 expression in combination with neural transmitters reveals better understanding of the developing ascidian brain

Rachel E. Holbert, Steven Q. Irvine

Bio. Sci. Dept., Univ. of Rhode Island, Kingston, RI, USA

Ciona intestinalis possesses a notochord and dorsal nerve cord during early development, evidence of its common ancestry with vertebrates. The ascidian brain is organized along the anterior–

posterior axis into a structure that resembles the vertebrate tripartite model, and includes the anterior sensory vesicle, neck region, and posterior visceral ganglion. A *Ci*-Pax6:GFP reporter gene has been transformed into wild type *C. intestinalis* embryos. At the late tailbud stage, fluorescent Pax6-expressing cells are visible in the sensory vesicle, visceral ganglion, and nerve cord. Visualization of neural *Ci*-Pax6 expression in combination with immunohistochemical fluorescence of domapinergic, serotonergic, and FMRF-amide expressing neurons provides an in-depth illustration of the developing ascidian brain. This work is complementary to ongoing studies of the effects of transcriptional inactivation of *Ci*-Pax6 on brain development in *C. intestinalis*. 12.00 Normal 0 false false false EN-US X-NONE X-NONE.

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Program/Abstract # 141

CLASPs in the *C. elegans* embryo

Eugenel B. Espiritu, Lori E. Krueger, Anna A. Ye, Lesilee S. Rose

Dept. of MCB, University of California, Davis, CA, USA

The *Caenorhabditis elegans* embryo undergoes a series of asymmetric cell divisions to produce daughter cells with different developmental fates. In the one-cell embryo, the centrosome–pronuclear complex centers and rotates to align with the anterior/posterior axis. At metaphase/anaphase, cortical pulling forces displace the spindle posteriorly, which results in unequal sized daughter cells after cytokinesis. Spindle positioning is regulated by PAR polarity cues and requires long astral microtubules to connect the spindle to the cortex, as well as dynein motor activity for cortical pulling forces. CLASPs, a family of microtubule plus-end associating proteins, rescue depolymerizing microtubules to promote long microtubules. *C. elegans* have three CLASPs: CLS-2, ZC84.3, and C07H6.3. CLS-2 is required for chromosome congression and central spindle formation, but ZC84.3 and C07H6.3 have yet to be characterized. To determine if CLASPs are required in spindle positioning, we knocked down CLASP expression using RNA interference. We confirmed that *cls-2* RNAi resulted in premature spindle pole separation, whereas there was no obvious phenotype after knockdown of ZC84.3, C07H6.3, or both. However, with RNAi of *cls-2* with either *zc84.3* or *c07h6.3*, some embryos exhibited failed pronuclear rotation and excessive posterior displacement of both spindle poles. Preliminary data suggests that this is a result of decreased astral microtubule length. Therefore, the three CLASPs may have a redundant function in regulating microtubule length. To further elucidate CLASP function, we are characterizing the sub-cellular localization of the CLASPs and plan to examine microtubule dynamics after *clasp* knockdown.

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Program/Abstract # 142

A pan-ectodermal enhancer module regulates the *Dlx-B* gene in *Ciona*

Steven Q. Irvine

Dept. of Biological Sciences, Univ. of Rhode Island, Kingston, RI, USA

The *CiDlx-B* gene is an early regulator of ectodermal development in the ascidian *Ciona intestinalis*. Sequence analysis of *CiDlx-B* reveals a 378 bp upstream region, termed B1, which is highly conserved with the corresponding region from the congener *Ciona savignyi*. The B1 element is necessary and sufficient to drive expression of a lacZ reporter gene in a pattern mimicking the early endogenous expression of *CiDlx-B*. This expression pattern which is specific to the entire animal hemisphere is activated preferentially in anterior, or a-lineage, cells by the distal